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1: Curr Mol Med. 2005 Mar;5(2):197-203.

Telomeres as biomarkers for ageing and age-related diseases.

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Telomeres in telomerase-negative cells shorten during DNA replication in vitro due to numerous causes including the inability of DNA polymerases to fully copy the lagging strand, DNA end processing and random damage, often caused by oxidative stress. Short telomeres activate replicative senescence, an irreversible cell cycle arrest. Thus, telomere length is an indicator of replicative history, of the probability of cell senescence, and of the cumulative history of oxidative stress. Telomeres in most human cells shorten during ageing in vivo as well, suggesting that telomere length could be a biomarker of ageing and age-related morbidity. There are two distinct possibilities: First, in a tissue-specific fashion, short telomeres might indicate senescence of (stem) cells, and this might contribute to age-related functional attenuation in this tissue. Second, short telomeres in one tissue might cause systemic effects or might simply indicate a history of high stress and damage in the individual and could thus act as risk markers for age-related disease residing in a completely different tissue. In recent years, data have been published to support both approaches, and we will review these. While they together paint a fairly promising picture, it needs to be pointed out that until now most of the evidence is correlative, that much of it comes from underpowered studies, and that causal evidence for essential pathways, for instance for the impact of cell senescence on tissue ageing in vivo, is still very weak.

Publication Types:

- Review
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